STN Search History

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=> d his

     (FILE 'HOME' ENTERED AT 08:17:37 ON 17 OCT 2002)

     INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
     DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 08:17:49 ON
     17 OCT 2002
               SEA (RSV OR (RESPIRATORY(A)SYNCYTIAL(A)VIRUS)) AND (M2 OR M2-1
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                1   FILE ADISALERTS
                1   FILE ADISINSIGHT
                5   FILE AGRICOLA
               78   FILE BIOSIS
               17   FILE BIOTECHABS
               17   FILE BIOTECHDS
               59   FILE BIOTECHNO
               10   FILE CABA
               17   FILE CANCERLIT
               99   FILE CAPLUS
                2   FILE CONFSCI
                8   FILE DDFU
              200   FILE DGENE
              278   FILE DRUGU
                1   FILE EMBAL
               71   FILE EMBASE
               51   FILE ESBIOBASE
                5   FILE FEDRIP
               24   FILE GENBANK
               11   FILE IFIPAT
               58   FILE LIFESCI
               65   FILE MEDLINE
                9   FILE PASCAL
                2   FILE PHARMAML
               10   FILE PROMT
               93   FILE SCISEARCH
               39   FILE TOXCENTER
              304   FILE USPATFULL
                5   FILE USPAT2
                1   FILE VETU
               44   FILE WPIDS
               44   FILE WPINDEX
L1               QUE (RSV OR (RESPIRATORY(A) SYNCYTIAL(A) VIRUS)) AND (M2 OR M2-
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     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, DGENE' ENTERED AT 08:27:41 ON 17
     OCT 2002
L2          513 S L1
L3          308 S L2 AND M2#####(P)(MUTA? OR SUBSTITUT? OR DELET? OR TRUNCAT? O
L4          230 DUP REM L3 (78 DUPLICATES REMOVED)
L5          223 S L4 AND (ANTIGENOME OR DNA OR RNA OR GENE  OR GENOME OR NUCLE
L6            3 S L5 AND CYSTEINE (S) M2?
L7          295 S L2 AND M2?(S)(MUTA? OR SUBSTITUT? OR DELET? OR TRUNCAT? OR FR
L8          228 DUP REM L7 (67 DUPLICATES REMOVED)
L9          221 S L8 AND (ANTIGENOME OR DNA OR RNA OR GENE OR GENOME OR NUCLEO
L10         198 S L9 AND (RSV (S) M2?)
L11          88 S L9 NOT PY>2000
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FILE 'HOME' ENTERED AT 08:17:37 ON 17 OCT 2002

=> index bioscience

L1   QUE (RSV OR (RESPIRATORY(A) SYNCYTIAL(A) VIRUS)) AND (M2 OR M2-1 OR M2(A)
         ORF-1 OR M2(A) 1)

=> d rank
F1          304   USPATFULL
F2          278   DRUGU
F3          200   DGENE
F4           99   CAPLUS
F5           93   SCISEARCH
F6           78   BIOSIS
F7           71   EMBASE
F8           65   MEDLINE
F9           59   BIOTECHNO
F10          58   LIFESCI
F11          51   ESBIOBASE
F12          44   WPIDS
F13          44   WPINDEX
F14          39   TOXCENTER
F15          24   GENBANK
F16          17   BIOTECHABS
F17          17   BIOTECHDS
F18          17   CANCERLIT
F19          11   IFIPAT
F20          10   CABA
F21          10   PROMT
F22           9   PASCAL
F23           8   DDFU
F24           5   AGRICOLA
F25           5   FEDRIP
F26           5   USPAT2
F27           2   CONFSCI
F28           2   PHARMAML
F29           1   ADISALERTS
F30           1   ADISINSIGHT
F31           1   EMBAL
F32           1   VETU

=> file f8,f2,caplus, biosis,embase,dgene
L2          513 L1
L3          308 L2 AND M2#####(P)(MUTA? OR SUBSTITUT? OR DELET? OR TRUNCAT? OR
                FRAGMENT)
L4           230 DUP REM L3 (78 DUPLICATES REMOVED)
L5          223 L4 AND (ANTIGENOME OR DNA OR RNA OR GENE OR GENOME OR NUCLEOTID
                E OR NUCLEIC)
L6            3 L5 AND CYSTEINE (S) M2?
L7          295 L2 AND M2?(S)(MUTA? OR SUBSTITUT? OR DELET? OR TRUNCAT? OR FRAGM
                ENT)
L8           228 DUP REM L7 (67 DUPLICATES REMOVED)
L9          221 L8 AND (ANTIGENOME OR DNA OR RNA OR GENE OR GENOME OR NUCLEOTID
                E OR NUCLEIC)
L10         198 L9 AND (RSV (S) M2?)
L11          88 L9 NOT PY>2000
```

=> d l11 2,4,5,11,15,16,18,30,88,86 bib,abs

L11 ANSWER 2 OF 88 MEDLINE
AN 2000387825 MEDLINE
DN 20304979 PubMed ID: 10846068
TI The Cys(3)-His(1) motif of the **respiratory syncytial virus M2-1** protein is essential for protein function.
AU Hardy R W; Wertz G W
CS Department of Microbiology, University of Alabama School of Medicine at Birmingham, 35294, USA.
NC AI12464 (NIAID)
AI20181 (NIAID)
SO JOURNAL OF VIROLOGY, (2000 Jul) 74 (13) 5880-5.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200008
ED Entered STN: 20000818
Last Updated on STN: 20000818
Entered Medline: 20000810
AB The **M2 gene** of respiratory syncytial (RS) virus has two open reading frames (ORFs). ORF1 encodes a 22-kDa protein termed **M2-1**. The **M2-1** protein contains a Cys(3)-His(1) motif (C-X(7)-C-X(5)-C-X(3)-H) near the amino terminus. This motif is conserved in all human, bovine, and ovine strains of RS virus. A similar motif found in the mammalian transcription factor Nup475 has been shown to bind zinc. The **M2-1** protein of human RS virus functions as a transcription factor which increases polymerase processivity, and it enhances readthrough of intergenic junctions during RS virus transcription, thereby acting as a transcription antiterminator. The **M2-1** protein also interacts with the nucleocapsid protein. We examined the effects of **mutations** of cysteine and histidine residues predicted to coordinate zinc in the Cys(3)-His(1) motif on transcription antitermination and N protein binding. We found that **mutating** the predicted zinc-coordinating residues, the cysteine residues at amino acid positions 7 and 15 and the histidine residue at position 25, prevented **M2-1** from enhancing transcriptional readthrough. In contrast, **mutations** of amino acids within this motif not predicted to coordinate zinc had no effect. **Mutations** of the predicted zinc-coordinating residues in the Cys(3)-His(1) motif also prevented **M2-1** from interacting with the nucleocapsid protein. One **mutation** of a noncoordinating residue in the motif which did not affect readthrough during transcription, E10G, prevented interaction with the nucleocapsid protein. This suggests that **M2-1** does not require interaction with the nucleocapsid protein in order to function during transcription. Analysis of the **M2-1** protein in reducing sodium dodecyl sulfate-polyacrylamide gels revealed two major forms distinguished by their mobilities. The slower migrating form was shown to be phosphorylated, whereas the faster migrating form was not. **Mutations** in the Cys(3)-His(1) motif caused a change in distribution of the **M2-1** protein from the slower to the faster migrating form. The data presented here show that the Cys(3)-His(1) motif of **M2-1** is essential for maintaining the functional integrity of the protein.

L11 ANSWER 4 OF 88 MEDLINE
AN 2000057898 MEDLINE

DN 20057898 PubMed ID: 10590093
TI **Respiratory syncytial virus** that lacks open reading frame 2 of the **M2 gene** (**M2**-2) has altered growth characteristics and is attenuated in rodents.
AU Jin H; Cheng X; Zhou H Z; Li S; Seddiqui A
CS Aviron, Mountain View, California 94043, USA.. hjin@aviron.com
SO JOURNAL OF VIROLOGY, (2000 Jan) 74 (1) 74-82.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000110
AB The **M2 gene** of **respiratory syncytial virus** (**RSV**) encodes two putative proteins: **M2**-1 and **M2**-2; both are believed to be involved in the **RNA** transcription or replication process. To understand the function of the **M2**-2 protein in virus replication, we **deleted** the majority of the **M2**-2 open reading frame from an infectious cDNA clone derived from the human **RSV** A2 strain. Transfection of HEp-2 cells with the cDNA clone containing the **M2**-2 **deletion**, together with plasmids that encoded the **RSV** N, P, and L proteins, produced a recombinant **RSV** that lacked the **M2**-2 protein (rA2DeltaM2-2). Recombinant virus rA2DeltaM2-2 was recovered and characterized. The levels of viral mRNA expression for 10 **RSV genes** examined were unchanged in cells infected with rA2DeltaM2-2, except that a shorter **M2** mRNA was detected. However, the ratio of viral genomic or antigenomic **RNA** to mRNA was reduced in rA2DeltaM2-2-infected cells. By use of an antibody directed against the bacterially expressed **M2**-2 protein, the putative **M2**-2 protein was detected in cells infected with wild-type **RSV** but not in cells infected with rA2DeltaM2-2. rA2DeltaM2-2 displayed a small-plaque morphology and grew much more slowly than wild-type **RSV** in HEp-2 cells. In infected Vero cells, rA2DeltaM2-2 exhibited very large syncytium formation compared to that of wild-type recombinant **RSV**. rA2DeltaM2-2 appeared to be a host range **mutant**, since it replicated poorly in HEp-2, HeLa, and MRC5 cells but replicated efficiently in Vero and LLC-MK2 cells. Replication of rA2DeltaM2-2 in the upper and lower respiratory tracts of mice and cotton rats was highly restricted. Despite its attenuated replication in rodents, rA2DeltaM2-2 was able to provide protection against challenge with wild-type **RSV** A2. The genotype and phenotype of the **M2**-2 **deletion mutant** were stably maintained after extensive in vitro passages. The attenuated phenotype of rA2DeltaM2-2 suggested that rA2DeltaM2-2 may be a potential candidate for use as a live attenuated vaccine.

L11 ANSWER 5 OF 88 MEDLINE
AN 1999318992 MEDLINE
DN 99318992 PubMed ID: 10388648
TI Support plasmids and support proteins required for recovery of recombinant **respiratory syncytial virus**.
AU Collins P L; Camargo E; Hill M G
CS Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, 7 Center Drive, MSC 0720, Bethesda, Maryland, 20892-0720, USA.. pcollins@atlas.niaid.nih.gov
SO VIROLOGY, (1999 Jul 5) 259 (2) 251-5.
Journal code: 0110674. ISSN: 0042-6822.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199907
ED Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990727

AB **Respiratory syncytial virus** (**RSV**) can be recovered from plasmids that separately encode antigenomic **RNA** and the N, P, L, and **M2-1** proteins of the nucleocapsid. However, in a recent study the inclusion of a separate **M2-1** expression plasmid was found to be unnecessary (H. Jin, D. Clarke, H. Zhou, X. Cheng, K. Coelingh, M. Bryant, and S. Li, Virology 1998, 251, 206-214). This suggested that the **M2-1** protein, which is a transcription antitermination factor, is not required to reconstitute the minimum unit of infectivity, namely a nucleocapsid fully functional for viral transcription and **RNA** replication. Here we show that the antigenomic plasmid is remarkably efficient as a **substitute** for an **M2-1** expression plasmid in supporting processive transcription by an **RSV** minigenome. Thus, the simple expedient of omitting an expression plasmid is invalid for evaluating recovery requirements. The issue of the requirement of **M2-1** for the recovery of infectious **RSV** is discussed.

L11 ANSWER 11 OF 88 MEDLINE
AN 1998371442 MEDLINE
DN 98371442 PubMed ID: 9705916
TI A single **nucleotide substitution** in the transcription start signal of the **M2 gene** of **respiratory syncytial virus** vaccine candidate cpts248/404 is the major determinant of the temperature-sensitive and attenuation phenotypes.
AU Whitehead S S; Firestone C Y; Collins P L; Murphy B R
CS Respiratory Viruses Section, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892-0720, USA.. sswhitehead@nih.gov
NC AI-000030 (NIAID)
AI-000087 (NIAID)
SO VIROLOGY, (1998 Aug 1) 247 (2) 232-9.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980827

AB **Respiratory syncytial virus** (**RSV**) cpts248/404 is a live-attenuated, temperature-sensitive (ts) vaccine candidate derived from cole-passaged cpRSV by two rounds of chemical **mutagenesis** and biological selection. Previous sequence analysis showed that these two steps introduced three single **nucleotide substitutions** into the cpRSV parent. Two of these occurred with the coding sequence for the L protein, and each resulted in a single amino acid **substitution**: Gin-831-Leu (248 **mutation**) and Asp-1183-Glu (404-L **mutation**). The third **mutation** resulted in a **nucleotide substitution** at position 9 of the c/s-acting **gene** start signal of the **M2 gene** (404-**M2 mutation**). In the present study, the genetic basis of attenuation of cpts248/404 was defined by the

introduction of each of these **mutations** (singly or in combination) into a full-length cDNA clone of cpRSV. Recombinant **RSV** derived from each **mutant** cDNA was analyzed to determine the contribution of each **mutation** to the ts and attenuation phenotypes of the virus. This analysis showed that the 248 **mutation** specifies a significant reduction of plaque formation at 38 degrees and is responsible for an intermediate level of attenuation in mice. In contrast, the 404-L **mutation** did not contribute to the ts or attenuation phenotype alone or in combination with other **mutations** and is thus an incidental change. unexpectedly, the 404-**M2 mutation** alone specified complete restriction of plaque formation at 37 degrees C an a high level of attenuation in mice. This indicates that the level of temperature sensitivity and attenuation of cpts248/404 can be attributed primarily to the 404-**M2 mutation**. Thus the cpts248/404 virus contains a set of ts and non-ts attenuating **mutations**, which likely accounts for its genetic stability. The recombinant version of this virus, rA2cp248/404, was phenotypically indistinguishable from cpts248/404 and represents a background into which additional **mutations** can be introduced as needed to obtain the desired level of attenuation for successful immunization of the very young human infant.

L11 ANSWER 15 OF 88 MEDLINE
AN 96102154 MEDLINE
DN 96102154 PubMed ID: 8524804
TI Production of infectious human **respiratory syncytial virus** from cloned cDNA confirms an essential role for the transcription elongation factor from the 5' proximal open reading frame of the **M2** mRNA in **gene** expression and provides a capability for vaccine development.
AU Collins P L; Hill M G; Camargo E; Grosfeld H; Chanock R M; Murphy B R
CS Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, MD 20892-0720, USA.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 Dec 5) 92 (25) 11563-7.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199601
ED Entered STN: 19960219
Last Updated on STN: 19960219
Entered Medline: 19960124
AB Infectious human **respiratory syncytial virus** (**RSV**) was produced by the intracellular coexpression of five plasmid-borne cDNAs. One cDNA encoded a complete positive-sense version of the **RSV genome** (corresponding to the replicative intermediate **RNA** or **antigenome**), and each of the other four encoded a separate **RSV** protein, namely, the major nucleocapsid N protein, the nucleocapsid P phosphoprotein, the major polymerase L protein, or the protein from the 5' proximal open reading frame of the **M2** mRNA [**M2**(ORF1)]. **RSV** was not produced if any of the five plasmids was omitted. The requirement for the **M2**(ORF1) protein is consistent with its recent identification as a transcription elongation factor and confirms its importance for **RSV gene** expression. It should thus be possible to introduce defined changes into infectious **RSV**. This should be useful for basic studies of **RSV** molecular biology and pathogenesis; in addition, there are immediate applications to the development of live attenuated vaccine strains bearing predetermined

defined attenuating **mutations**.

L11 ANSWER 16 OF 88 CAPLUS COPYRIGHT 2002 ACS
AN 1999:223017 CAPLUS
DN 130:263124
TI attenuated recombinant **respiratory syncytial virus** expression systems and vaccines
IN Jin, Hong; Tang, Roderick; Li, Shengqiang; Bryant, Marty
PA Aviron, Inc., USA
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9915631	A1	19990401	WO 1998-US20230	19980928
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2304932	AA	19990401	CA 1998-2304932	19980928
AU 9895852	A1	19990412	AU 1998-95852	19980928
EP 1017791	A1	20000712	EP 1998-949553	19980928
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
PRAI US 1997-60153P	P	19970926		
US 1998-84133P	P	19980504		
US 1998-89207P	P	19980612		
WO 1998-US20230	W	19980928		

AB The present invention relates to genetically engineered recombinant RS viruses and viral vectors which contain heterologous **genes** for use as vaccines. In accordance with the present invention, the recombinant RS viral vectors and viruses are engineered to contain heterologous **genes**, including **genes** of other viruses, pathogens, cellular **genes**, tumor antigens, or to encode combinations of **genes** from different strains of **RSV**.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 18 OF 88 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1992:368318 BIOSIS
DN BA94:50368
TI SEQUENCE AND IN-VITRO EXPRESSION OF THE **M2 GENE** OF TURKEY RHINOTRACHEITIS PNEUMOVIRUS.
AU YU Q; DAVIS P J; BROWN T D K; CAVANAGH D
CS DIV. MOL. BIOL., INST. ANIM. HEALTH, HOUGHTON LAB., HUNTINGDON, CAMBRIDGESHIRE PE17 2DA, UK.
SO J GEN VIROL, (1992) 73 (6), 1355-1363.
CODEN: JGVIAY. ISSN: 0022-1317.
FS BA; OLD
LA English
AB Negative-stranded virion **RNA** and oligonucleotide primers complementary to fusion (F) protein **gene** sequences were used to generate cDNA clones, revealing that the **gene** 5'-proximal to the F protein corresponded to the **M2** (22K) **gene**, as in respiratory syncytial (RS) virus. The transcription start signal,

GGGACAAGU, was identical to that of the F and matrix (M) proteins to turkey rhinotracheitis virus (TRTV). There were two sequences with the potential to function as transcription termination/poly(A) signals, located at **nucleotides** 751 to 762 and 777 to 787; 15 clones derived from mRNA indicated that the first of these sequences formed the major signal. Part of the next downstream (5') **gene** was sequenced; unlike mammalian pneumoviruses the TRTV **M2** **gene** did not overlap the beginning of the 5'-proximal **gene** . Northern blotting indicated that infected Vero cells contained less **M2** mRNA than F mRNA and that about half of the **M2** mRNA was present as a F-**M2** dicistronic mRNA. The **M2** **gene** contained two overlapping open reading frames (ORFs 1 and 2), as with RV virus. ORF 1 comprised 558 **nucleotides** with the coding potential for a 186 amino acid polypeptide Mr, 20959, eight or nine residues shorter than for human RS virus strains. The overall amino acid identity was 40%, the N-terminal one-third of the proteins sharing 62% of residues, the remainder 29%. A hydropathy plot of the TRTV **M2** protein had close similarity to that of the **M2** of RS virus. The protein was predicted to have a basic character with no N-terminal signal sequence or other major highly hydrophobic sequences. In vitro translation of a transcript comprising both ORFs 1 and 2 produced a single product of apparent Mr 23,000, corresponding to the **M2** product of ORF 1. Site-directed **mutagenesis** confirmed that this product was derived from ORF 1 and that frameshifting was not involved. The second ORF was expressed only from a transcript which lacked the AUG codons of ORF 1 and, although occupying a similar position to that in the RS virus **M2 gene**, had vitually no amino acid identity in its 73 residue length and was approximately 25% shorter than the corresponding RS virus ORF 2. The hydropathy plot of the potential products of the second ORFs of TRTV and RS virus showed little resemblance. Taken together these results suggest that ORF 2 is unlikely to be expressed in vivo. Our accumulated data show that TRTV has the partial **gene** order 3' M-F-**M2** 5', whereas the corresponding RS virus **genes** are arranged 3' M-SH-G-F-M 25'.

L11 ANSWER 30 OF 88 DGENE (C) 2002 THOMSON DERWENT
AN AAX35042 DNA DGENE
TI Recombinant **respiratory syncytial viruses**
IN Bryant M; Jin H; Li S; Tang R
PA (AVIR-N) AVIRON INC.
PI WO 9915631 A1 19990401 85p
AI WO 1998-US20230 19980928
PRAI US 1998-89207 19980612
US 1997-60153 19970926
US 1998-84133 19980504
DT Patent
LA English
OS 1999-244413 [20]
AN AAX35042 DNA DGENE
AB The specification describes recombinant **respiratory syncytial virus** (**RSV**) particles and viral vectors which express heterologous **genes** or **mutated** **RSV genes**. The **RSV** particles comprise a **RSV antigenome** or **genome** containing at least one functional **deletion** in an **M2 gene**, or encode antigenic polypeptides of both **RSV**-A and **RSV** -B, or contain a L **gene mutation**. The recombinant **RSV** particles can be used to produce vaccines, e.g. bivalent vaccine against **RSV**-A and **RSV**-B, or **RSV** and influenza. Recombinant **RSV** vaccines can also be constructed for viruses such as HIV-1, HIV-2 and HBV, by constructing a **RSV**

comprising a heterologous sequence from these organisms. The present oligonucleotide was used to construct the ribozyme/T7 terminator sequence, which was construct vectors which are used in the course of the invention.

L11 ANSWER 88 OF 88 DGENE (C) 2002 THOMSON DERWENT
AN AAQ25033 cDNA DGENE
TI Bovine **respiratory syncytial virus** **genes** - used in the prodn. of agents for use in detection and as vaccines for BRSV infection.
IN Samal S K
PA (SAMA-I) SAMAL S K.
PI WO 9207940 A 19920514 74p
AI WO 1991-US8177 19911104
PRAI US 1990-608937 19901105
DT Patent
LA English
OS 1992-183675 [22]
AN AAQ25033 cDNA DGENE
AB This is the entire genomic sequence of bovine **respiratory syncytial virus** (BRSV) strain AAA51908 and contains the N, P, M, SH, G, F, **M2**, and 3' L **genes**. The BRSV **nucleotide** sequence and **fragments** of it may be used for the detection of BRSV infection, partic. in cattle. The encoded BRSV proteins can be used to detect BRSV antibodies and as vaccines to prevent infection. The proteins can also be used for the prodn. of antibodies. The sequence was obtd. from viral mRNA extracted from BRSV-infected MDBK cells by the guanidine thiocyanate-CsCl method, followed by 2 cycles of oligo-(dT)-cellulose column chromatography. See also AAQ25034 and AAQ25035.

L11 ANSWER 86 OF 88 DGENE (C) 2002 THOMSON DERWENT
AN AAV17553 cDNA DGENE
TI Attenuated **respiratory syncytial virus** ~~vaccines - useful to protect individuals against **RSV** infection~~
IN Bukreyev A A; Collins P L; Juhasz K; Murphy B R; Teng M N; Whitehead S S
PA (USSH) US DEPT HEALTH & HUMAN SERVICES.
PI WO 9802530 A1 19980122 238p
AI WO 1997-US12269 19970715
PRAI US 1997-47634 19970523
US 1996-21773 19960715
US 1997-46141 19970509
DT Patent
LA English
OS 1998-110579 [10]
AN AAV17553 cDNA DGENE
AB This is the 5'-3' positive sequence **nucleotide** sequence of **respiratory syncytial virus** (**RSV**) D46. The **genome** is negative-sense; the complete **nucleotide** sequence of the wild-type B-1 virus has also been determined (see AAV17552). A novel infectious recombinant **RSV** comprises a **RSV genome** or **antigenome**, a major nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large polymerase protein (L), and a **RNA** polymerase elongation factor, where the recombinant **RSV** has at least two attenuating **mutations**, one of the **mutations** specifying a temperature-sensitive (ts) **substitution** at amino acid Phe521, Gln831, Met1169 or Tyr1321 in the **RSV** polymerase **gene** or a ts **nucleotide substitution** in the **gene** -start sequence of **gene M2**. Also claimed are: (1) an isolated infectious **RSV** particle which comprises a recombinant

RSV (anti)**genome**, N, P, and L proteins, a **RNA** polymerase elongation factor, where the (anti)**genome** is modified: (i) to ablate or modulate expression of a SH, NS1, NS2 or G **gene** or a cis-acting regulatory sequence; and (ii) by a termination codon introduced within a selected **gene**, or by a change in sequence, position or presence of a GS or GE transcription signal relative to the selected **gene**; (2) an expression vector; and (3) an **RSV** strain selected from cpts **RSV** 248 (ATCC VR 2450), cpts 248/404 (ATCC VR 2454), cpts 248/955 (ATCC VR 2453), cpts **RSV** 530 (ATCC VR 2452), cpts 530/1009 (ATCC VR 2451) or cpts 530/1030 (ATCC VR 2455), or B-1 cp52/2B5 (ATCC VR 2542) or B-1 cp-23 (ATCC VR). The isolated attenuated recombinant **RSV** and **RSV** particles are used in a vaccine to stimulate the immune system of an individual to induce protection against **RSV**. The expression vector of (2) is used for the production of infectious attenuated **RSV** particles.